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MONOLATERAL CONGENITAL COLOBOMA OF THE IRIS

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This case report is submitted for two reasons: First, to present a somewhat classic form of coloboma of the iris, except for the fact that it here occurred only in one eye, and second, to show the value of stereoscopic photography in reporting any condition of the anterior section of the eye.

Case report: Mrs. A. J., 38 years of age, referred by local hospital for refraction.

Present history: C. C. Blurred distant vision. No previous Rx. Occasional frontal headaches. Diplopia when viewing lights (?) observed for past six months. Moderate epiphora.

Remote history: Patient illiterate, foreigner, no previous eye examination; suffers from gall-bladder trouble, and chronic constipation. Anaemic and underweight. Familial history indicated that one sister living in Europe has congenital coloboma of both irises. No other member of family is so affected.

External examination: Right eye: Pupil of medium size. Pupillary reflex normal to light both direct and consensual, as well as to convergence. Versions normal in all meridians. Cornea clear, aqueous chamber of normal depth. Left eye: "The pupil as a whole is displaced downward, so that its upper border lies approximately behind the center of the cornea. It is continued below into the coloboma and is hence pear-shaped" (Fuchs). The photograph indicates a slight projection of iris tissue from the nasal side into the coloboma. This, if continued to the opposite edge of the pupil, forms what is known as a "bridge coloboma." Examination by the Gullstrand Slit Lamp at

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the Clinic showed this projection to extend further than is indicated in the photograph. It is extended almost halfway across the coloboma.

Ophthalmoscopy: Right and left eyes were negative—no coloboma of any other part was visible in the left eye.

Refraction: O. U. —7.50 D. S. to R. 20/40 L. 20/40.

Summary: A case of monolateral congenital coloboma of the iris presented itself for refraction. All findings were non-pathologic. Refraction prescribed—muscle balance not investigated—stereoscopic photography of iris ordered and here presented.

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THE ETIOLOGY AND TREATMENT OF STRABISMUS*

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History

It would be utterly impossible in the few short hours which we have, to enter very deeply into the subject of strabismus or into the numerous ramifications and discussions of the subjects so closely related to it. You have previously discussed the anatomy of the extrinsic ocular muscles, the pathology of the orbit and its contents, the physiology of vision and the principles of psychology underlying it. So we will make no attempt to discuss them and temporarily overlook (if such a thing is possible) these allied subjects, subjects which no doubt play a great part in the Etiology of Strabismus.

In order that we may get the true background for the work, let us take up a subject which to most of you will be dry and uninteresting but which is really necessary in order that we may get the right perspective on the subject at hand. We will follow a short resume of the history of the work on strabismus.

Hippocrates was the first whom we know of, who took note of squinting children or at least gave us any definite writing on the subject, however slight it may have been. He was the first to note the presence of a paralytic type as differentiated from comitant; he also noted the part which heredity played in its development. From Hippocrates to Paulus Aegueta there were several men who noted the anomaly, but Aegueta was the first to put forth serious effort to find a treatment for it. This he accomplished by placing a mask over the face with two small perforations before the eyes, believing that to look through the holes, the eyes must necessarily straighten.

It was not, however, until the first part of the eighteenth century that any considerable notice was given to squint. It was in this period that many of the superstitions, as they may be called, arose, e. g., that squinting was due to the influence of evil spirits or that it was produced by imitating other people—at this time also originated many ideas concerning strabismus which are still held today.

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During this period lived a Dr. Taylor, oculist to the King of England, about whose methods little is known, but who is given credit for originating the idea of blocking off the fixing eye so that the squinting eye would fix.

Nothing of great importance was done along this line until after Tenon in 1806 had written extensively on the Anatomy of the Orbit.

The first real tonotomy is credited to Diffenbach of Berlin who performed the operation in 1839. Closely following this, there were developed numerous techniques for the work and later Von Graefe developed the advancement operation. Following this "plague of cutting" as Parinaud called it, Donders presented his accommodation theory which was eagerly accepted by many of the foremost ophthalmologists of the day. And finally, in summing up the work of many other authorities and previous inclinations toward it, Claude Worth of London, in 1903 brought out the fusion theory, the theory which probably holds more followers today than any of the others, especially is this true in this country.

Etiology

In taking up the etiology of strabismus we are confronted with a problem which has over a hundred answers and yet has no answer. Like many pathological conditions of the body, the true or exact cause seems indeterminable. It is true that while there have been numerous theories as to the etiology of squint, there are only three which have stood the test of time. At the present time we are working under a new theory which, while it combines the substance of the rest, certainly makes a step farther in the eventual solution of the problem, if there ever will be one.

Let us first take up the three outstanding theories up to the present, outlining briefly the merits and demerits of each.

The muscular theory was outstanding during the first part of the eighteenth century. It was believed, by the adherents of this theory, that comitant squint was due entirely to the increased strength of one of the rectii muscles over its antagonist. Due to its shortness or a peculiar insertion, it pulled the eye nasalward or laterally as the case might be. As a result of this theory we have tonotomy and the operation of advancement which is linked to it. There is no question but what this theory has merit but the results of the fallacies of it, are numerous.

The accommodation theory was first described by De La Hare and elaborated upon by Von Grafe and Helmholtz before Donders became its sponsor and to whom it is credited. In this theory we have our first real establishment of the relationship of accommodation and convergence; Donders claiming that most convergent squints were due to "the disturbance of the relationship between accommodation and convergence" caused by hyperopia and hyperopic

astigmia and following, that most divergent squints were a direct result of myopia, and as proof of this point, had corrected the refraction of numerous squint cases with astonishing results. But still there were those cases which did not yield to this treatment or to the former treatment of tonotomy. There is no question but what the work of Donders was the first real step to the solution of the problem, if there ever will be one. All of you have treated the refractive squint case, as we call it today, and have seen the strabismus disappear, apparently, until the lenses have been removed.

Stevens strongly opposes Donders' theory, stating that we have no anatomical or neurological basis for it. Stating also that recent research has definitely proven that the nucleae controlling accommodation is distinctly separated from that governing convergence and that the nerve fibers pass out of the brain separately and only unite in a common sheath after considerable space. He also claims that the relationship between the two, accommodation and convergence, is purely a matter of habit and is not organic as Donders supposed, as they can readily be disassociated. There is research being done along this line now, at the University of Southern California and whether or not there is a definite organic relationship remains to be seen, though Stevens is authoritative.

The fusion theory which was first suggested by Javal and Parinaud was finally established by Worth and up until recently has held in this country at least, a majority of followers and it must be remembered that to those not practicing surgery this theory is the sweetest.

Fusion is the simultaneous blending of the two retinal images into one mental picture and necessitates the following prerequisites: We must have two healthy maculae, a set of perfectly co-ordinated ocular muscles, goverened by a complex nervous mechanism capable of adjusting them to a nicety. In studying the mechanism of single binocular vision we are getting into the field of physiology and neurology which we intended to keep away from if possible, but these faculties interlace so closely that it is almost an impossibility to talk about one without bringing in the other. Let us take up the practical application of the fusion theory. According to Worth, the fusion sense is normally developed by the sixth month after birth and well advanced by the twelfth and so definitely fixed by the end of the sixth year that nothing but an actual paralysis can cause the eye to deviate. However, in the progress of development, there are numerous things which may retard or permanently impair the fusion faculty:

1. Hyperopia.
2. Anisometropia.
3. High latent lateral or vertical phorias.

4. Specific fevers—those affecting accommodation.
5. Injury at birth.
6. Violent mental disorders—extreme fright or anger.
7. Heredity.

Worth's theory has a great many followers and just as many who argue that if there is a true fusion sense it would not seem logical that it could be trained in so short a time as two or three months and hold for the rest of a lifetime. Also that it would seem that poor fusion was the result, rather than the contributing factor of strabismus, due to the fact that most squints only appear after two or three years. Also that in some cases where there is a high amblyopia present, there is often a fair grade of fusion. It is to these disbelievers of Worth's fusion theory that we owe the theory upon which we are working at the present and which personally seems the more logical. While many have had the same concept in recent years, I believe that Wilkinson is the first to state definitely the modern trend of thought.

The nervous theory is based on two premises: quoting from Wilkinson:

"1. Pathological Nervous Lesions.

Under which heading we have those retinal disturbances such as detachment, coloboma, lesion resulting from tabes, retinitis and any other causing loss of the foveal guide.

Next we have muscle paralysis, both of the rectii and the ciliary.

The presence of scotoma, relative or absolute, any of which reduce vision below 6/6.

These latter according to Lloyd, are the direct causes of numerous cases of squint, which is perfectly true, but as he continues his discourse he gives changes of the visual field as a contributing cause and here I believe he is confounding the cause with the effect for although we find definite field changes in most cases of strabismus, it does not necessarily mean that it is this that has caused the squint.

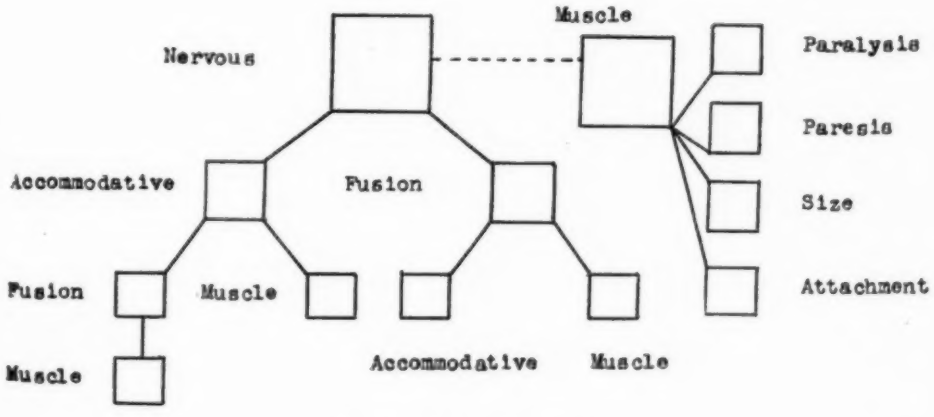
2. Functional Nervous Manifestations.

This is summed up very efficiently by Harmon as a lesion or defect of the cerebrum involving a weakness or possible absence of that most human desire for binocular vision—as the basis for manifest squint."

The statement which Harmon gives here is really nothing but a summation of Worth's fusion theory and I believe we could put into a more apt statement. Let us say the strabismus is an ocular anomaly concerning apparent deviations from normal of the visual axis, and while having numerous contributing causes, is primarily an neurophysiological abnormality resulting in an organic change.

In discussing the nervous theory, remember we are not trying to fix dogmatically the cause for squint, but are endeavoring to find some reasonable basis for those obstruse cases of strabismus in which there is no anatomical muscle change, in which there is little or no refractive error, in which there can be elicited a good grade of fusion, but, in which there seems to be a chronic suppression. Especially does this theory stand forth in cases of falsely alternating squint which have passed, so to speak, all the requirements of the other theories and yet fail to straighten. It may be interesting to note that in experiments now being carried on, using hypnotism and suggestion, we are endeavoring to break down chronic cases of suppression. If successful, the nervous theory will certainly take its place with the others.

In treating strabismus we must start at the end and work backwards, taking but one step at a time. If there is a morbid change in the muscles themselves we must first rectify this condition before trying to build up any grade of ocular co-ordination. Let us liken this procedure to a man rebuilding his house after a fire. Does he start his new foundation on the ashes of the old? No, he first clears away the debris and starts on a new and solid basis. So with making practical our nervous theory, we must first, by operation or otherwise, clear away all morbid changes and then start to rebuild, breaking down any existing neurological resistance and building up by training or teaching a new neural path for ocular control. Whether this control is to be put to any one use as accommodation, convergence or fusion or put to the combined uses of them all, depends entirely upon the primary diagnosis.



R. M. Hall. Figure 1.

In summing up these four theories of the etiology of strabismus,

there is no question but what each has its place, the magnitude and intensity of each depending upon the individual case. That some squints have for their basis a true muscular anomaly is apparent. That some are purely refractive is true. That some high phorias and true constant squints are due to an impaired fusion faculty or to direct retinal or cerebral pathology is also understood. But coming within the scope of true optometric practice are those cases of strabismus, whether latent or manifest, due to any one or a combination of the above causes, which need orthoptic training. This does not, in the modern sense, mean a training of the fusion sense but rather of the systematical building up of the synaptical reflexes or of the breaking down of the resistance offered to them. In teaching, as would a teacher of the piano, new habits of mental procedure, it means a training of ocular co-ordination, of fixation and projection and the assimilation of all of these to one end, the overcoming of the disfigurement.

Contributing Causes of Strabismus

There are numerous possibilities that, while there has been no direct connection made by writers, on the etiology of squint, contribute to the cause of squint. In looking over this list, and adding a hundred and one others to it from your personal experience, you can readily understand the wide disagreement and variation in the theories of strabismus. Remember that these may be but the contributing factor in one squint case and do not hold as a general basis. Chiefly they are as follows:

1. Hypermetropia.
2. Heterophoria.
3. Anisometropia.
4. Amblyopia.
5. Acute illness.
6. Injury at birth.
7. Heredity.

Wilkinson and Stevens stress the point of declination as a probable cause but in the vast majority of cases we have found that declination is secondary. Stevens also points out that the shape of the orbit has much to do about many of the cases but here again it seems improbable as the percentage of squint cases seems to have no relation to percentage of noticeably deformed eyeballs and orbits.

However, the part which hypermetropia plays in squint is well recognized, although that part is not as large as Donders believed it. Due to the linked relationship of accommodation and convergence, the hyperope finds it necessary to accommodate in excess for the near point and it thus follows that he will over converge, in order that he may secure comfort, one eye fixes the near object, while the other eye turns in, taking up the excess convergence of the two

eyes. So if the relationship of these two functions held rigidly, we have most of our hyperopic patients beginning to squint, but we know this is not so and we have found that the relationship between the two can be broken up.

It seems that giving heterophoria as a cause of squint is out of order, so to speak, for while the phoria is no doubt a precurser of the manifest variety, it is in itself a type of strabismus and the cause of the phoria is probably at the bottom of the difficulty in the manifest squint.

Logically and clinically, anisometropia is one of the most important factors in squint. One of our prerequisites for good single binocular vision is a refractive media capable of focusing objects clearly on the maculae. But here we have two greatly dissimilar images on the macular areas and the urge to see singly is lessened, if not lost entirely.

Amblyopia is another essential cause, as it interferes with clear, retinal stimulation. In the majority of cases, however, I believe that the amblyopia is acquired after the eye has begun to squint and certainly it is greatly increased. The amblyopic condition we find in cases which have had the refractive error corrected, is certainly an element to be considered. Injuries at birth and corneal scars which may form in a few hours after birth are certainly contributing factors; especially the paralysis of the lateral rectii muscles which is altogether too frequent.

Heredity plays an important part, probably the most important. You all know of families of squinters and Worth's 51.78% of all squints being traced to heredity, speaks for itself.

Before going into the various developments and treatments for a strabismic case, it is essential that at this time, in order that we may get the subject well in mind, we take up a differential classification of the types encountered and the methods used to disclose them. Omitting for the present the vertical tropias, we find that in the main all lateral squints divide themselves into two wide classifications, namely, convergent and divergent. To enter into a discussion on the widespread nomenclature used in various parts of the world, would be a waste of time and take a good sized volume in itself, so let us confine ourselves to the terminology used at present in this country.

Classification of Types

A. Convergent.

1. Paralytic or con-comitant.
2. Unilateral or bi-lateral.
3. Periodic (occasional) or constant.
4. Alternating.

a. True (essential).
b. False (accidental).

5. Spurious or pseudo.

B. Divergent.

1. Paralytic or comitant.
2. Unilateral or bi-lateral.
3. Periodic (occasional) or constant.
4. Alternating.
   a. True (essential).
   b. False (accidental).
5. Neuropathic.

C. Vertical.

1. Paralytic or comitant.
2. Unilateral or bi-lateral.
3. Periodic (occasional) or constant.
4. Alternating.
   a. True (essential).
   b. False (accidental).

As you can see from the outline, we have five different classes into which our convergent types may fall, namely, paralytic or non-paralytic, periodic or occasional, unilateral or bi-lateral, alternating (either true or false) and pseudo. The same is true of the divergent cases except that our fifth type is neuropathic. The differentiation of each type and class we will discuss more fully under diagnosis, as what is said about one class will hold, with few exceptions, to all classes.

A point I wish to stress particularly is this: that all the techniques, instruments and perspiration in the world will not help one bit, if the original diagnosis is wrong. No doubt many of you wondered why we should stress the classification of the types of strabismus so strongly, and that is the reason. Practitioners wonder why they are not getting the results they should. In plain language, they have diagnosed the case wrong in the first place. It is imperative that, before any treatment can be outlined, we have the correct diagnosis, not one which is a snap judgment but one which is made after keeping the patient under observation for at least a month or longer. Even then, there is doubt as to whether fusion training will bear results and it is only until the case is completed, that you really can be sure.

The Methods Employed

Of the numerous tests used for diagnosing strabismus, I will outline briefly only those which are easily made and are being used in every-day practice. With one or two exceptions, these tests may

be used to give us a pretty thorough understanding of most squint cases.

History

At this point I wish to stress the importance of taking a searching history and of the imperativeness of an accurate diagnosis. It is essential that we note every detail in the history, for it is here that we learn of the duration of the squint, the approximate time of its appearance, the progress it has made up to this time and the reasons given for its cause. Now going back: we learn from a careful questioning of the parent or of the patient himself, whether the squint is periodical or constant. Many of you will say that you can learn that from the screen test, but we cannot with any degree of certainty, because there are times when the deviation will be more noticeable than at others, and unless you have the patient under almost constant observation, it is impossible to detect it. Parents will come with the complaint, "Well, Johnny's only cross when he is real tired or when he coughs hard." This is one of the big points in the successful diagnosis of an occasional squint. A constant squint of high degree is easily discernible from observation but it is the low deviation which tests the skill of the diagnostician. We also make use of the screen test in the differentiation of unilateral and bi-lateral strabismus which is our next division of squints. The use of complimentary colors for the detection of tropias of this type is a test really worth while, for here we get a true test, while in the screen test care must be used not to overlook facial deformities, bulbophthalmus, narrow canthae, etc.

Inspection

Simple inspection most frequently reveals the presence of a squint (either convergent or divergent), the location of the fixing eye and also the probable degree of the deviation. Also from inspection we may learn other facts which are of great value. Tilting of the head in heterotropia, especially those which are paralytic, ptosis and many little idiosyncrasies which the squinter uses to get relief and to conceal his disfigurement.

Corneal Image Test

By checking carefully the position of the corneal images, as produced by the reflected ophthalmoscope light, we can detect any asymmetry. Normally, with the subject fixing the peep hole in the ophthalmoscope, the corneal images will appear about 1 or 1½ mm. to the nasal side of the optic axis. Wider or narrower angles indicate strabismus or variation of angle gamma.

Screen Test

The screen or cover test as it is sometimes called, is probably the most simple, yet the most universally used check for phorias

and tropias we have. While not accurate to a fineness it is elastic and greatly facilitates the diagnosis. The patient is instructed to fix the target either distant or near; the fixing eye is then occluded; if any deviation is present in the other eye, it must then deviate out or in to fix. If the redress is in, the squint is divergent and vice versa. Care must be exercised however, in using the screen test, due to the simulation of divergent squint where there is a high hypermetropia with its corresponding wide angle gamma and also not be deceived by a high phoria. The screen test, while seemingly simple, demands experience on the part of the operator to readily differentiate between phorias, tropias and variations in the angle gamma.

Diagnosis

We have already discussed the importance of accurate history taken in connection with strabismic cases and of strabismus, so let us now take up the diagnosis of concomitant strabismus.

We have in the main, three methods at our disposal, which if followed rigidly, will expose the presence or absence of a paralytic squint. The first which is more or less a subjective technique, is the presence of a persistent diplopia. It is necessary that the examiner question the patient closely and minutely, calling his attention to different highly colored objects about the room, as the false image is often so faint as to be almost negligible. In older patients, the diplopia a common complaint. In patients too young to speak, this method is obviously useless. The second test, and probably the best for all classes of patients, is the version test. With one eye occluded at a time, the patient is told to follow the light or fixation object as the operator moves it from side to side and up and down. If the eye fails to follow the light and stops with a jerky motion, there is a paralysis or paresis on that side (which may include one or more muscles). There is one thing which must be watched carefully and that is the similarity between poor ocular rotation and paresis. In poor rotation, the eye must be coaxed, but will move slowly. The third, and the one which is more commonly used today, is the detection of an insistent diplopia under complementary red and green lenses. The patient faces the white object, one eye being covered with a red lense, the other being covered with a green, which is complementary to the red; as the object is moved from side to side, the diplopia will increase or decrease, increasing on the affected side. This method is useless on very young patients and necessitates the patient keeping his head perfectly still.

For diagnosis of periodic squint whether convergent or divergent, we depend almost entirely upon the case history, both from the close questioning of the parents and the patients themselves. It is the type most usually affected by ocular fatigue, especially the

element of fusion or of reflex convergence (third grade convergence). There is generally no squint on arising in the morning but as the eyes tire, the squint appears. Its manifestation is due in great part to the diminishing strength of the fusional reflex, oftimes in conjunction with a slight amblyopia. These squints must not be confused with those of the accommodation type, where a latent or manifest hyperopia causes an over-accommodation and consequent over-convergence at the near point. In the true sense of the word, periodic or occasional squint is fusion apathy, or a temporary weakening of the negative fusional reserve. There are writers today who say that there is no such fusional element as a negative reserve. Maybe they are right, but here is one case that will give them something to explain, if there isn't.

Worth gives us the classification of neuropathic divergent strabismus and we are certainly in need of some broad division into which to put those difficult cases of divergence which we find only too often. The divergence nearly always dates back to infancy. It may be constant or occasional, unilateral or alternating. There is seldom a good fusion faculty present if any at all. There is scarcely any refractive error, although sometimes when the squint is of long standing, an amblyopia will be found. The deviation is constantly varying. Most of these individuals have enough dynamic convergence to bring both eyes into play at the near point, but seem to lack the urge to use it. They are of a nervous, high strung character and resent any reference to their deformity. In many of these cases we get a history of epilepsy and insanity. In this class too must go those women, who after the menopause, become highly exophoric or tropic.

Parallelling this class but not in it, are those exotropes resulting from progressive myopia and blindness. Needless to say, operation is the only sure cure in these cases.

And now we come to the one stumbling block of all squint—alternating strabismus. In your studies and your reading you will find volume on volume written on the diagnosis, prognosis and treatment of every type of tropia imaginable, but start looking for good, authoritative reading on alternating strabismus and if you can find two consecutive paragraphs you are lucky. More about this later. The terminology used to designate the different types of alternating strabismus is sadly deceptive and I believe a thorough understanding of the terms used in this type of squint is necessary before going very deeply into the subject. According to Worth, we have two classifications: (a) Accidentally alternating—those which alternate because the refraction and other conditions are very similar in both eyes, and (b) Essentially alternating—in which there is little refractive error but in which there is congenital loss of the fusion cen-

ters. It would seem to me that the first type might be called a false alternation as it can be relieved by non-operative treatment and the second type, Worth's essentially alternating, the true type, because congenital adversity to fusion necessitates operative procedure. The diagnosis of an alternating squint can be had from the screen test, but the differentiation between true and false alternation takes a long, complicated procedure.

Besides the numerous types of squints mentioned and the thousand and one not mentioned, there are two which are common enough to be spoken of at this time. The first, the spurious types found in infants and due presumably, to gastric disturbances or slow development of the fusion faculties and a pseudo strabismus, caused by failure of the nasal canthae to properly open up, due to slow facial development.

Non-Pathological Anomalies of Strabismus

We have of course, any number of pathological anomalies which bear directly on the diagnosis, treatment and prognosis of strabismic cases; however, there also are many non-pathological or we might say, physical anomalies, which are of main importance to the student of myology. Symptoms which are in no way related to pathology and symptoms which, if overlooked, ofttimes cause failure in the case under question.

False Maculae

Probably the most important of these and the one most serious to deal with, is the case of false maculae. Here we have a false, sensitive area, a definite physical change, taking place as the squint is becoming constant and necessitating immediate attention, if treatment is to be successful. This spot has no real increase of sensitivity but merely retains what sensitiveness it had previous to the deviation, while the surrounding area and real macular area has become amblyopic. Also in most of these cases it will be found that the brain has learned to make allowance for the faulty position and projects the image to the spot of direct fixation. In some cases where a tenotomy does not fully correct the error, we sometimes get a triplopia, due to a faulty projection, by the patient, of the image of the strabismic eye. Here we have projection of the true fovea, the false fovea and the intermediate sensitive spot. Treatment of false maculae lies almost wholly in the hands of the ophthalmic surgeon and consists of straightening the deviating eye, followed by persistent blocking off of the good eye and then finishing with fusion training. However, the optometrist now has ways open to him which although arduous, are oftimes successful. The non-deviating eye is securely patched and a pinhole placed centrally over the deviating eye. It will be found that the eye is more sensitive to one color or the others, of red, green, blue or yellow, then a slightly

reduced correction is given the poor eye combined with the color to which the eye is more susceptible and the patient is made to wear this, two or three hours a day. This procedure may be altered with a stenopaic slit placed vertical, and combined with the ordinary training. Thankfully, true false maculae is comparatively rare and care must be exercised not to confuse this true condition with a faulty sense of fixation.

Faulty Fixation

Faulty fixation is found in most squint cases of long standing and in many strabismic cases just beginning to squint. The "wandering eye" as it was called, because of the fact that when the good eye occluded the deviating eye would not come to direct fixation but wander in any meridian or at any angle, is not due necessarily to an amblyopic condition but is also found where there is poor ocular rotation or control. It is also clearly noted in infants who have not as yet developed the fusion sense. True fixation is easily established in most cases by simply blocking off the non-deviating eye for a short time; sometimes, however, it is found necessary to coax the eye to get rotation before a definite fixation can be obtained.

Faulty Projection

Another anomaly which is often confused with the former is a faulty projection which sometimes follows paralysis or a paresis of the rectii muscles, or pathological disturbances of the retinal elements, such as detachment or thrombosis of the retinal arteries or orbital tumors. While this anomaly is due to pathology, it in itself. is not essentially pathological but due more to experience (faulty), as the mind has learned that it is much more comfortable to project the image to the point of fixation. Time alone can remedy this condition.

Strabismometry

Quoting Wilkinson: "The importance of the accurate determination of the degree of squint cannot be too strongly impressed—as an aid in determining the type and etiology of the squint and a guide to the future treatment."

As almost every author has his own method of determining the degree of a squint, it would be impossible to describe all, but I will try to give the ones in common use and the method of procedure.

Lawrence's strabismometer consists of a flat piece of ivory, concave on one side to fit the lower lid and scaled to the right and left from the center. The good eye is occluded and the squinting eye fixes, then the strabismometer is placed beneath it and the eye uncovered, the squinting eye again deviating, the degree of which can then be measured. The inaccuracy of this method is obvious.

Other methods have been devised by Hirschenberg, Javal, Char-

pentier, Maddox, Priestly Smith and others. The two methods of the most accuracy and the quickest to use are Maddox's tangent strabismometer and Priestly Smith's tape. In using Maddox's method, the patient is placed facing a light at the meter distance and fixing on the tangent scale; the operator is seated facing the patient between him and the scale. Watching the corneal images, the operator points out the figures of the tangent scale and approximating the degree of the squint tells the patient to look at that figure. If, when upon seeing it, there has been no addition movement, that is the true amount of the squint. The Priestly Smith method is approximately the same, but more accurate.

The myoculator also proves of value in the measurement of strabismus. The patient is instructed to fix a light placed in the center of the screen and the movable target is so placed that the corneal images are the same, the amount of deviation being the number of degrees found necessary to move the target.

Development of a Typical Case of Convergent Squint

"An esophoria may disappear if the conditions causing it are abolished or offset. Thus an accommodative convergence-excess usually disappears if the casual hyperopia and astigmatism are corrected early enough and it disappears in presbyopes who no longer accommodate excessively because they cannot. Again, in some cases a convergent squint disappears in youth, because in the growth of the face, the orbits get more divergent so that the interni work with more difficulty than before" (Weiss).

"In other cases an esophoria increases. This is seen in the *development of a convergent squint,* which usually begins as an accommodative convergence-excess. The steps in this development are as follows:

(1) A child with a good deal of hyperopia or astigmatism, as soon as he begins to use the eyes much for near work, develops a varying, evidently more or less spasmodic esophoria (convergence-spasm, see supra) which is marked for near when his convergence is called into play, but which is little or not at all marked for distance.

(2) Little by little, the deviation increases until binocular fixation for near becomes impossible, and the periodic esophoria is transformed into a periodic *convergent squint.* This, at first is intermittent and occasional, afterwards more and more constant. There is now more or less esophoria for distance also, although always less than for near, and there is still binocular fixation for distance.

"Careful tests at this stage will show in every case that *the squint is associated with diplopia.*

(3) After this condition has lasted for a long time, perhaps some years, the patient gives up binocular fixation for distance, so that he has now a convergent squint for both distance and near

(continuous squint). The deviation as measured by the screen, however, is still much more marked for near than for distance. Diplopia can still usually be elicited by the tests. The condition is still mainly one of convergence-excess.

(4) Later still, the amount of deviation for distance begins to increase until ultimately the squint for distance equals that for near points, and both are absolutely greater than they were. This change is due to the super-adding of the divergence insufficiency to the primary convergence-excess; the conditions now in fact, are those shown in the table as obtaining for a convergence-excess and divergence-insufficiency combined. The rotation of the eyes is still normal in amount, indicating that the muscles themselves are as yet unaffected.

"At this stage diplopia, if it can be elicited at all, no longer obtrudes itself upon the patient's notice nor causes confusion. In many cases it cannot be elicited by any tests, suppression of the images having occurred.

(5) Later, secondary muscular changes (contracture of the internus, stretching and weakening of the externus) take place in the squinting eye, producing an absolute reduction in the rotation outward and excess of rotation inward.

"In some cases it seems that a *convergent squint may develop from a divergence-insufficiency*. The steps in this case are, first, an increase of the esophoria for distance, then the super-adding of a convergence-excess producing an esophoria for near. The result is, first a periodic convergent squint (esotropia for distance, esophoria with binocular fixation for near); later a continuous squint for all ranges. Until late in their course, such cases are distinguished from those starting with a primitive convergence-excess by the fact that the deviation for distance always exceeds that for near."

Procedure

We have at our disposal at present, an ever-increasing number of techniques, as we might call them, for use in orthoptic training. While the instrumentation of each is different, the underlying principles are the same and the end for which all strive is identical. To say that the end is arrived at by the use of any one of these methods, is probably untrue; but by the combined use of all, each doing that particular type of work for which it has proven fit, non-operative treatment has unquestionably increased its effectivity.

It must be thoroughly understood that there can be no rigid steps or procedure set down as to treatment of any individual case of strabismus. The treatment of any case depending entirely upon the circumstances met with and the facilities which the operator has to overcome them. However, we can roughly outline a general procedure which may be of some value:

(1) Correction of refractive errors.
(2) Reduction of the amblyopia.
(3) Establishment of true projection.
(4) Establishment of good ocular rotation and co-ordination.
(5) Development of the fusion faculty.
(6) Re-establishment of the reserves.

1. *Correction.*

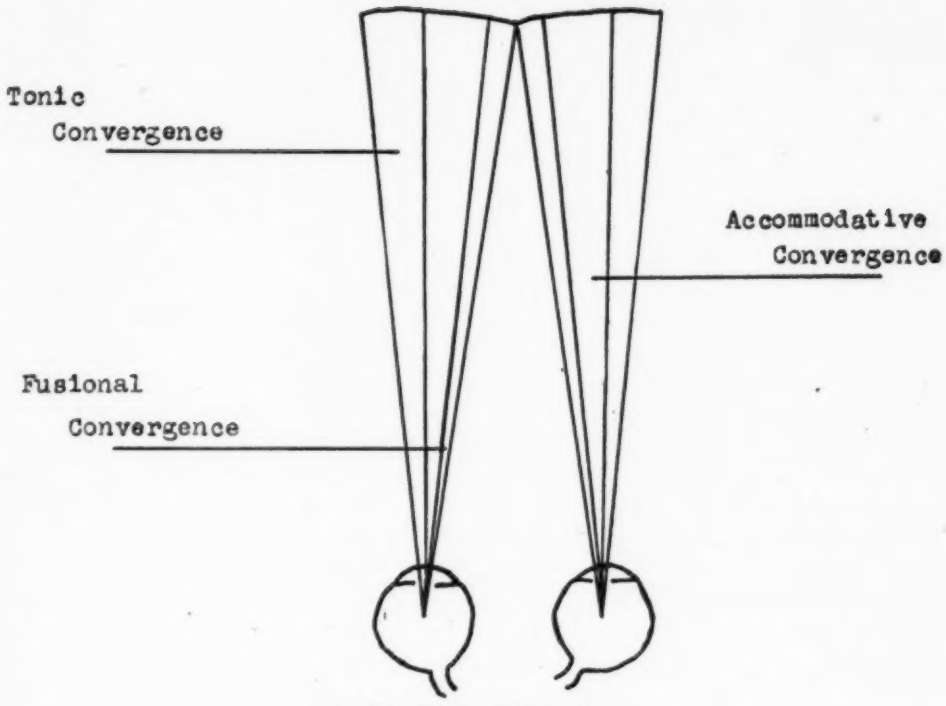
As we stated in studying the different etiological factors governing strabismus, hyperopia or hyperopic astigmia left uncorrected, there necessarily is an increased effort of accommodation together with an associated convergence. While the relationship between the two is not absolute or unbreakable, it is strong enough so that any great amount of interference will cause diplopia and eventually a squint. Hence we can readily see the absolute necessity of keeping the total refractive error corrected at all times.

It will be well to, in so much as many of our so-called refractive squints are relieved by the practical application of these theories, that we study the writings of Howe, Worth, Maddox and others on the subject of convergence. Remember that we are just running over these things hurriedly to freshen up a few points which you may be applying in your refraction but which you have a tendency to drop in working with squint.

Here you will see diagrammed (Fig. 2) the three grades of convergence as outlined by Maddox. The tonic convergence is that tested when we use the Maddox Rod test at 6m and it is this test that determines the amount of divergence—insufficiency or excess. We also have here a certain amount of reflex convergence.

Added to this as we approach the near point, we have the convergence due to accommodative action, which we measure by first introducing a vertical doubling prism. Following Maddox, we find that for every diopter of accommodation there is about three-quarters of a meter angle of convergence. Therefore it follows that in hyperopia we usually will find an accommodative convergence-excess. Hence the necessity for a full dynamic correction in all cases of refractive squint.

Also, we have as a third element, fusional or reflex convergence, which according to Sheard, is the most affected by ocular fatigue. It is to this element we turn in eliminating squints of the periodic type and also in effecting a sufficient reserve to keep any pair of eyes straight. Many squints are directly attributable to a low fusional convergence, even in cases where there is no over-accommodative action. Many of the so-called muscular squints are probably due more to the low nervicity reserve of the fusion centers or to the high resistance offered to it by anisometropia than to any true muscular insufficiency or mal-attachment.



R. M. Hall. Figure 2.

2. *Reduction of Amblyopia.*

After the refraction has been carefully checked, we may proceed to the elimination of the amblyopia (if present). It is essential here, as in the preliminary diagnosis, that the operator know exactly just what type of amblyopia he has to deal with. If it is refractive, the proper Rx worn for at least a month, should begin to bring results. If the amblyopia is toxic, the only thing possible is the removal of the toxin. If it is an exanopsia, a strict routine must be laid down for the patient to follow; systematic occlusion of the good eye, pinhole spectacles, office training with the amblyoscope, etc. When upon using the full correction upon the squinting eye and fully blocking off the good eye both for distance and near, over a period of three or four weeks, the vision of the deviating eye does not come down to at least 20/60, we must take care, for we have then either an amblyopia exanopsia of long standing or a congenital difficulty. To differentiate between the two is difficult and probably at the best our methods are none too accurate. With the good eye occluded, we hold two similar targets before the deviating eye; if while the patient fixes on the one immediately in front of him and

yet pronounces the one on either side to be seen more clearly, we judge that it is a case of exanopsia. But if both objects appear equally blurred, the diagnosis would be that of congenital amblyopia. Of course, there are some cases of high refractive error remaining uncorrected over a period of years, which will not yield to any test or treatment and those cases also of high astigmatic error which have been corrected, the axis of the cylinder placed in the wrong position, which often gives a simulated amblyopia.

Clinically you will probably note that the reduction of amblyopia is not constant but seems to reduce in a series of steps (see Fig. 3). Following is a graph of the time periods taken in twenty-five cases, all giving vision less than 6/90. You will note that reducing the vision from 6/90 to 6/30 took almost as long as the reduction from 6/30 to 6/12 and also the length of time taken to reduce below 6/12.

All amblyopic cases are doubtful and after considerable time has been allowed for the return of function with little or no improvement, then the case must be operated and will at the best get only an aesthetic result.

3. *Establishing True Projection.*

Projection, rotation and co-ordination, while important factors, are easy to re-establish but require time and patience. I believe the myoculator proves itself best for this purpose because of the moving targets and the psychological effect it has on the squinter. A large spot is projected on the screen and the patient is told to keep his finger on it as it moves in the different meridians. Rotation is better established if each eye is taken separately.

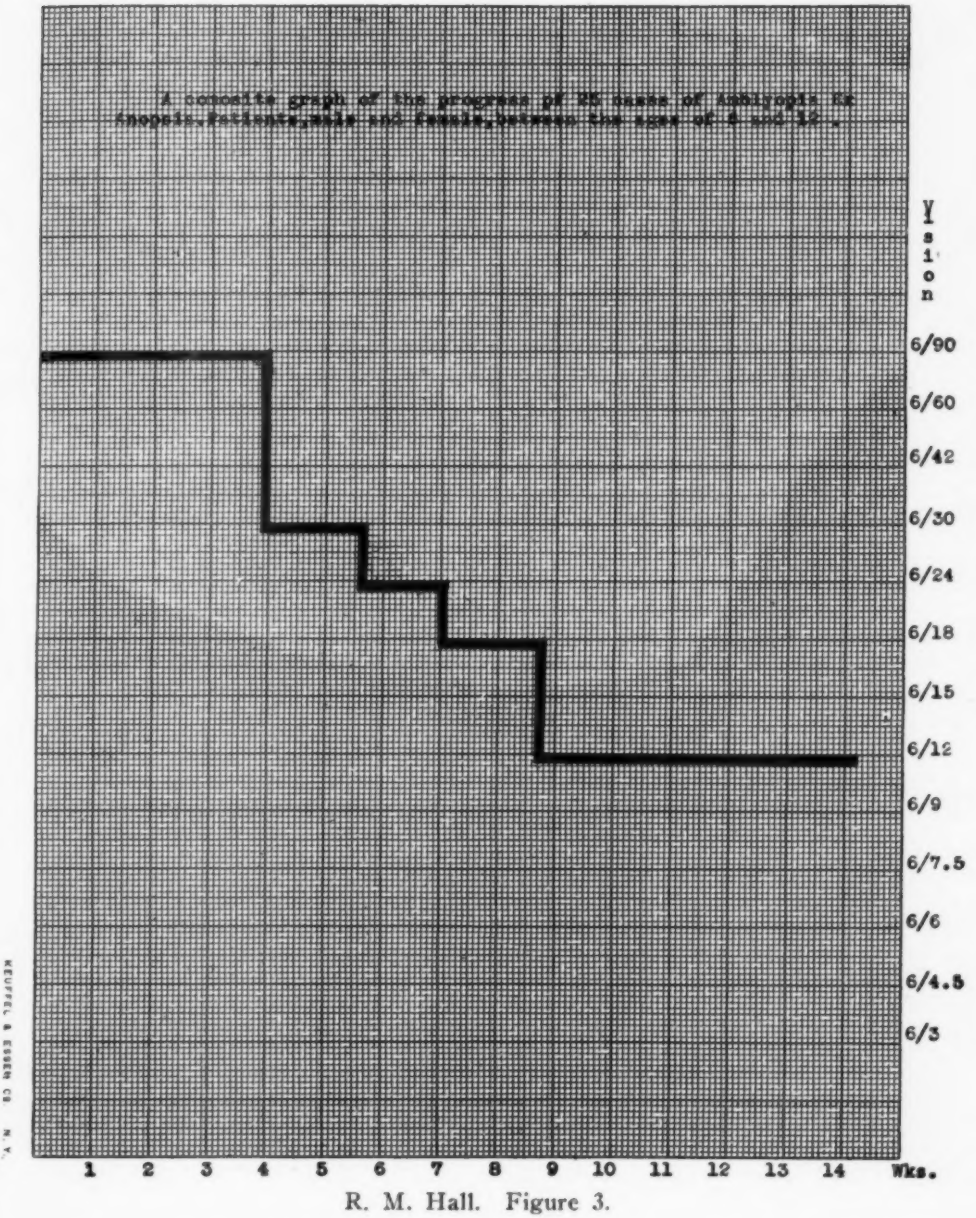
4. *Rotation and Co-ordination.*

Again I believe the myoculator is the most expedient in getting good ocular rotation and co-ordination. We can establish good free action in any meridian and at any distance by simply adjusting our screen and carefully instructing the patient.

We have said previously that in reducing a squint we must first bring that squint back, by a reversion of the processes with which it was developed, to its beginning. And we find most generally that when the morbid muscle changes have been corrected, the amblyopia reduced, the ocular rotation and fixation re-established and ammetropia corrected, that we have left the squint in its primary stage, each eye seeing well, functioning properly in itself, but used alternately. Maybe this alternation is not noticeable, it may be only a transference of vision, nevertheless there is that alternation present.

And so this brings us to the subject of alternating strabismus; its diagnosis, classification and treatment.

You will find upon going through the literature on squint, chapter after chapter on every type of squint except alternating. Of all the literature only a total of four full pages are devoted to this



R. M. Hall. Figure 3.

most important subject, and of these four only two paragraphs are pertinent. The first by Maddox in which he states, "the only cure is operation." The second by Sheard is the only place in literature which sounds the importance of this stage of squint.

"In alternating strabismus where first one eye and then the other is used, each eye is able to maintain its vision and power unimpaired. Nature certainly points the way in this decided manner and it seems wisdom, therefore, to transfer every case of fixed unilateral squint into an artificially alternating one by exclusion of the good eye for several hours each day and compelling it to assume the squint for that period."

In this short paragraph Dr. Sheard has sounded the keynote of all work on strabismus. He has summed up chapter after chapter of theory into a statement which is as practical as simple addition. This is the true basis for non-operative treatment of squint.

In the main, we have two primary classifications of alternating, namely those of Worth:

(a) Essential—where there is little or no refractive error and vision is perfect in both. However there is a congenital adversity to fusion. Fusion training is of no avail as there is a total absence of the fusion centers.

(b) Accidental—where the refractive error is the same in both eyes. If they remain without treatment, one eye usually becomes dominant and the squint becomes unilateral.

If you will recall our primary classification, you will see that the nomenclature is slightly different. Worth's essential being termed *True* and his accidental termed *False*. While a change of nomenclature is not necessary, it seems to clarify the types.

On the market recently have appeared many new and startling *machines* for the purpose of straightening cross-eyes; these instruments are capable of doing everything except that; however, while they are theoretically unsound and logically incorrect, they prove of value (each in its own way) in aiding in non-operative treatment.

If Dr. Sheard is correct in his statement and I more firmly believe he is as I go along, the myoculator, the kratometer and others are of little or no value in breaking down alternating strabismus as such. I am cognizant of the fact that certain techniques have been developed to care for this certain type and that they do a great deal of good in many cases but from a practical standpoint I am a firm believer in the amblyoscope.

We have discussed the diagnosis of alternating strabismus as differentiated from the other types and have taken up the difference between the two main types found and so now our problem is to find a suitable method of differentiating between the two clinically and also a suitable instrumentation for the treatment.

There is only one way which I know of to differentiate between the two and that is to use the amblyoscope, and by using the amblyoscope, I mean to make a thorough, painstaking search of the case in

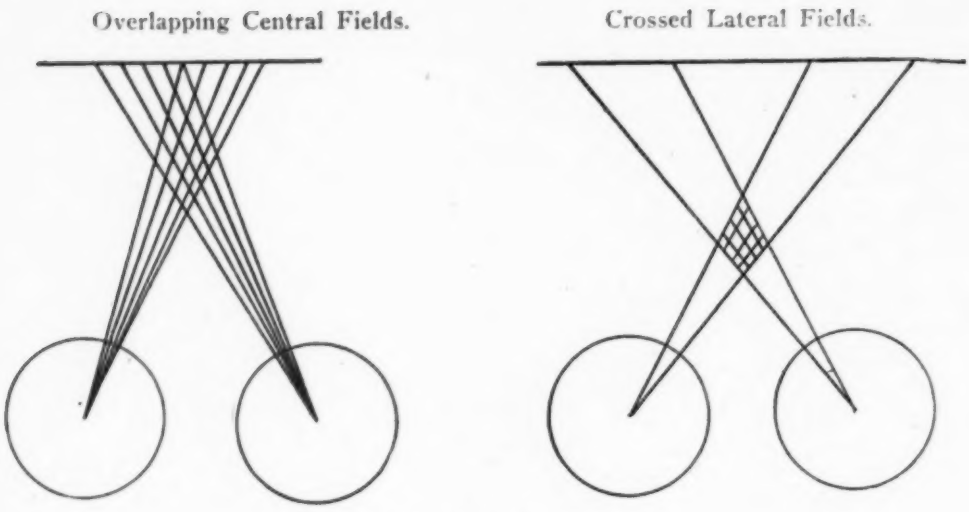
order that we may gain a thorough knowledge of the fusion faculty. If we have done our work right up to this time, we should have no trouble in eliciting simultaneous binocular vision, especially in the crossed lateral fields. However, in cases where the alternation is of long standing, it oftimes takes two months to produce it. In cases of true alternating squint you will find that simultaneous binocular vision may be elicited in the crossed lateral fields only, while in those cases which are false, you will find that single binocular vision and fusion can be established in the overlapping central fields but often takes considerable time.

In my personal practice, I try for one month (12 to 15 minute daily periods) to break down the overlapping fields. It is usually easier to elicit simultaneous binocular vision in crossed fields, even in cases of true alternating squint, than to get single simultaneous binocular vision in the overlapping central fields of a false alternation (see Fig. 4).

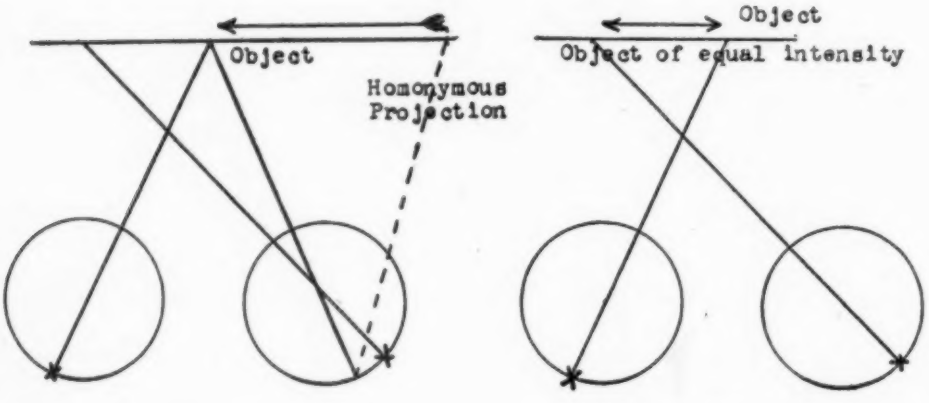
As to the treatment of true alternating strabismus, we have no optometric means and in reality neither has the surgeon, for most of these cases only revert to convergency or become divergent after a time, due to total lack of the fusion reflex.

In treating strabismus in the falsely alternating stage, we do not follow the same procedure as we do in squints or phoria of the non-alternating type, but work in an altogether different manner. Previously with convergent cases as with esophoria, we have been endeavoring by prisms or orthoptic training to bring the homonymous false image (kratometer, myoculator and stereoscope techniques) nearer to the true image. To do this we have used prisms (base-out) to displace, or to actually turn the eye outwards (by the myoculator). Now we are endeavoring to build an image, or rather a separate heteronymous object equal in intensity and proportions to the real object only on or within range of the visual axis of the deviating eye. In other words, we have a true crossing of the visual axis with a simulated heteronymous diplopia, e. g., see Fig. 5.

I have found and I believe your findings will bear me out that until single simultaneous binocular vision is obtained with the amblyoscope, other instrumentation is of little value. It is true that the same technique may be applied to the myoculator and the kratometer but only by combining them first with the Well's technique. I am not condemning these instruments, so please do not misunderstand me, but I do believe and sincerely that the amblyoscope is preferable in this instance, due to the variety of object control. After good, single simultaneous binocular vision has been obtained with the amblyoscope within a central overlapping field of not over 15 degrees, the work is greatly facilitated by the use of the other instruments.



R. M. Hall. Figure 4.



I. Convergent Unilateral.
(a) Myoculator.
(b) Wells' Technic.
(c) Kratometer.

II. Alternating Convergent.
(a) Amblyoscope.
(b) Rotary Prisms.

R. M. Hall. Figure 5.

After binocular vision is well established, we may start to develop true fusion, of bringing the two images closer together (this is aphoria now and we are decreasing it) until they are fused into one. Not a mechanical matching of images, for then there is chance for the return of suppression, but true fusion, preferably the fusion of complementary colors. The myoculator technique seems to facilitate matters a great deal at this point and give us, at the

same time, true fusion and amplitude, as well as stereopsis. This method, I believe, is the more preferable due to the elasticity and great variation of angle.

5, 6. *Development of Fusion Faculty and Re-establishment of Reserves*

However, our job of fusional development has only started at this point; from here we must build a solid and lasting foundation as it were. Many squints will still be apparent, even at this stage (another argument against Worth's fusion theory) and we have a real task before us in building up the reserves, or rather of breaking down the resistance which is still offered to single binocular vision. And here modern ingenuity has helped us greatly, in giving us the kratometer and the Well's technique. I believe that for this type of work they cannot be surpassed. Certainly I know that any one of these instruments which we have been discussing is supposed to do the whole routine from producing binocular vision to turning out a pair of spectacles for the patient to wear, but they will not work. However, each has its place and used there, with slight variations, are a great aid to our ultimate end. The kratometer of Hazen and the stereo-phorometer of Well's were both developed for the same purpose (fusional training) neither obtaining their end in its entirety but both becoming a handy tool as we might say, in the re-establishing of the reserves but both working from opposite directions. In using the kratometer, we make use of the shock technique, by increasing the prismatic resistance, so-called, in jumps of two, three, five or more prism diopters at once; where in the Well's technique we use a very gradual increase. Both have their merits and both their faults, but as the Well's technique is probably the newer to us, I will endeavor to outline briefly its use. Going directly to reserve building, the easiest fusion distance is found by using the B-1 chart, which consists of six round, black discs, numbered beneath from three to eight, separated from a single disc over which there is an arrow and by use of prism power base in or out, the arrow is brought over the number six disc. This is done as the objects on all the cards are separated by a distance of 6 cm. always remembering to make any prism change while the eyes are closed. The same procedure is followed on B-2 chart for vertical deviations. Then using the "E" series of charts (pails, tubs, tubes, etc.) we test for perspective. Testing for suppression we use series "F" (words with control markings) and so on until we start actual work on the reserves, for which we use series "C" (On NE). For esotropia we use the widest P. D. possible with the least amount of prism power, which gives fusion and gradually reduce the P. D. until the letter "N" breaks or doubles, the patient then closes his eyes and the P. D. is again widened, gradually the prism base-out may be

reduced and as the reserve is built, prism base-in may be substituted. The procedure is the same for exotropia except that the P. D. is from narrow to wide. Of course this is but a rough outline and the workings of the technique, while quite simple, are interesting.

In trying to reestablish fusion after we have brought the squint back into its alternating stage, we meet with many curious types. Some, especially those of divergence, respond readily to fusion training. Some, those which have had a high degree of convergence, but which has been reduced to about one-third its original deviation, will be able to get single binocular vision in practically all fields except that within the remaining third of the squint. The regional relationships of a pair of eyes of this order are equalized in all but a small lateral area. This is probably the main reason why many squints, while upon quick observation show a complete recovery, under a careful inspection, will show a slight strabismic cast.

Another type of case is that in which, by our various procedures, we have managed to straighten and which can pass successfully 90% of the tests made for fusion; including binocular mixing of colors, perception of depth, etc., but has still remaining a strong rivalry-relation between the eyes which cannot overcome the suppression. This can probably be explained by the fact that during our so-called fusion training, we have set up secondary corresponding points within the macular area, which are equally sensitive to the points of primary correspondence.

Other cases which we meet are those called "Congenital loss of the fusion sense" according to Worth. But holding more to our nervous theory, they are those cases in which there is so great a rivalry-relation between the two eyes that there is seemingly a true adversion to any grade of binocular vision. However, this condition may be present in cases other than those that squint.

All of these odd cases are strong arguments for Stuart's condemnation of Worth's fusion theory as Worth only assumes that there is a definite anatomical fusion center existing. These cases tend to strengthen the belief that binocular vision is not in the true sense binocular at all by control, but an acquired habit—rather than a true physiological function.

Another point which I wish to stress emphatically is the misuse of the words, suppression and suspenopsia. It is true that in squints of recent standing where there is an annoyance by persistent diplopia, the squinter will wilfully and consciously suppress the false image. True suspenopsia has no place in optometry, as it is really a pathological condition caused by hysteria, glaucoma, cerebral and retinal hemorrhages. The word which most writers intend to use, instead of these two, is transference.

A similar condition exists in alternating strabismus, except that

we have an actual physical deviation of the globe combined with the visual energy flow. There is no subconscious suppression or suspension in the so-called suspenopsia cases, it is but a change of nerve flow over another set of neural archways entirely, called upon by a conscious desire for point fixation.

I am fully cognizant of the fact that such a belief overthrows our theory of fusion centers; however, I believe that if you stop and study you will find that to hold to the nervous theory will necessitate only a change of nomenclature and application as the techniques and results from either the fusion concept or the nervous concept are the same.

And now to return to our other instrumentation such as the stereoscope and reading bar. These do not need to be taken up at any length due to their common use. Both have a distinct value in home training for which they are well suited and easily applied.

The Stereo-Campimeter and Its Relation to Strabismus

It would seem that we should have taken the campimeter up under the general head of instrumentation, but as long as its position is comparatively new in connection with this line of work, I believe that in discussing it by itself, we will get a more comprehensive view of its usefulness.

The primary help from the campimeter comes in the detection of central and para-central scotoma. It is obvious that with either of these present, orthoptic training is impossible.

On the other hand, let us take a case where there is no scotoma present; we can map the fields at first and from time to time after that and note the actual progress of our work by the ever increasing size of the fields.

As to the shape and size of the fields and their relation to a phoria or tropia; we find that true heterophorias will constrict the fields in one meridian only and that the constriction is readily overcome when the phoria is eliminated. Paresis of an extrinsic muscle will not, however, cause a constriction while a true paralysis will. Tropias give us a picture of a decentered constriction with an enlarged area on the side of the squint.

Diverging for a minute into a correlated field, the study of phorias, we can also learn much of benefit to us from the campimetric fields. In the type of cases which we come across frequently, where we find a great variation of duction power, low in the morning and gradually increasing as the day goes on, we will note, upon taking the color fields along with the other refractive findings and symptoms, a toxic condition, usually stimulative. Needless to say, any effort to build the duction power or even train the fusion faculty, while this condition is present, will result in failure.

Note: Discuss the cause of convergent strabismus in myopia—according to Hansen and Grut this is due to sensation of distance.

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AN OPAQUE HYALOID CANAL*

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A young woman, 37 years old, presenting some very common symptoms of eyestrain, including conjunctivitis, came to me for an examination a few weeks ago. She had always supposed that her vision was normal in both eyes until a short time ago when she applied for a position with a railroad. She was found to have such poor visual acuity with the right eye that she barely succeeded in obtaining employment.

Retinoscopic examination showed only a slight hyperopic error—the reflex through the left pupil was quite clear but the right was clouded as if there was pus in the anterior chamber.

Ophthalmoscopic examination revealed an opaque attachment spreading out against the capsule at the posterior pole of the lens. Beginning with a plus sixteen lens in the ophthalmoscope it appeared not unlike the funnel of a cyclone—very black at the attachment and growing fainter and smaller as it retreated into the vitreous. As this was brought into focus by reducing the lens power it became black at points farther and farther back into the vitreous, until with a plus eight its corkscrew course had been traced nearly to the disc. It was impossible to get a sharp focus further.

This patient was sent to her physician for treatments to clear up the rather bad conjunctivitis combined with eczema of the right lid, advised that help for her right eye was very unlikely, and told to report for an examination later.

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STRABISMUS AND AMBLYOPIA

At a recent meeting of the West and Northwest of London and of Middlesex I. O. O. Local Association, the entire meeting was devoted to the presentation of case reports dealing with the correction of amblyopia and strabismus by optometric means. In this important work, in a large sense, rests the future of optometry, as it is by successfully treating these difficult cases that the prestige of optometry is enhanced.

The following report by F. G. Hawkes, fellow of the British Optical Association, is typical of those which were presented and indicative of the splendid work which is being done by optometrists in England:

R. G., aet. 11½, seen March 2nd, 1931. Medical examination at school revealed left eye amblyopic. Parents had no knowledge of it, but boy stated that he told his mother two weeks previously he could not see so well with his left eye as with his right. No attention was paid to this remark. Father wears strong plus cylinders in both eyes. Mother emmetropic. Fundus examination normal. Retinoscopy showed R. + 1.0. L. + 1.0 + 4.00 axis 100°. Subjective test: Right eye V. A. 6/6+1.0 D. S.=6/6. Left eye V. A. 6/60+1.0 ⊃ + 3.00 D. Cyl. axis 100° = 6/24 —1. P. D. 60 mm. Cover test showed slight divergence of visual axes. Red and green light test disclosed suppres-

sion of left eye. Given shade for right eye the patient was told to approach total occlusion as nearly as possible in view of school requirements. Exercises for distant type and block letter printing suggested. Told to report on the 9th inst.

March 9th, 1931. Examined result of writing exercises, separation points very marked. Told to continue these exercises, striving to improve all the time. Series II. Pictures. To encourage amblyopic eye, 7.5 cms. separation. Blended with 16$^{\Delta}$ base out. Stereogram too wide but fusion held to 6$^{\Delta}$ base out = 10$^{\Delta}$ abduction. Details picked up fairly well with left eye. 6 cm. card blended at 14$^{\Delta}$ B. at 7$^{\Delta}$ R. at 12$^{\Delta}$.

March 17th, 1931. Amblyopia tested, having worn shade but not constantly. V. A. = 6/24.

Series 1., 6 cms. Double at zero; 8$^{\Delta}$ base out to blend carries to 4$^{\Delta}$ (slowly moving prisms). L. controls mark (bottom) begins to move outwards. Recovers at 8$^{\Delta}$, carries to 16$^{\Delta}$. R. at 10$^{\Delta}$. Thought best to concentrate on amblyopic series IV. Half hour training, gradually appreciating that left eye is being encouraged to dominate under stimulus of stronger appeal. Continued up to change over point only. Finally, V. A. test showed 6/18.

March 18th, 1931. Continued with amblyopic series. Can manage up to change-over, but finds difficulty with good eye favored. Shade not worn more than one quarter of day. Told must wear all day. Wrote letter to school to this effect. Bandaged eye with pad and plaster.

March 20th, 1931. Refraction done again. Find that + 4.0 D. Cyl. axis 100° gives one letter of 6/12. Continued amblyopic series sheet No. 7. Can distinguish center lines as separate near the dots but following along, fixation, indifferent, so interprets as one faint blurred line. Very encouraging sign showing gradually awakening perception under guidance and stimulus. Last figure central dot seen faintly. All above exercises with 3.75$^{\Delta}$ base out.

March 25th, 1931. Given new prescription. Left eye at end of visit reads all but two letters of 6/12 line. Still exercise with amblyopic series. Perceives more readily. Amblyopia gradually becoming less deep.

March 30th, 1931. Reports that eyeshade is no inconvenience now, but notices everything duller than with right eye open. Amblyopic series now accomplished with ease. As a change used synoptophore with minstrel slide. Fused at zero; carried to 2½° div. 15° convergent. Fusion faulty but little impaired. Small type reading, J. 2 can now be appreciated; notices that print appears blacker.

April 8th, 1931. Writing exercises over Easter show improvement. V. A. now 6/12 slowly. Effort to fix very noticeable. Letters lose position, showing macula not completely restored to sensitivity.

April 9th, 1931. Red and green test and cover test reveal parallel axes. Pencil test fair up to 1 in. sep. at 10 ft.

Stereo slides 21 and 22 correct, all but 5 and 7 miscalled. Same mistake on reversing.

Minstrel slide. 7° deviation; 40° convergence.

Spot light. 1° better each way.

V. A. 6/12 slowly, but can now read J. 1 slowly at 33 cms.

April 13th, 1931. V. A. 6/9 — 3. J. 1 well. Stereo slides correctly named.

April 15th, 1931. Refraction, right eye, 6/6 + 0.75 ⊂ + 0.25 axis 165 = 6/6, but can still read 6/6 with + 1.50 D. S. + 2.0 D. Sph. blurs to 6/12. Left eye, + 4.50 D. Cyl. axis 105° = 6/9 — 3. Note: Muscle test, ordinary way, reveals 1° exophoria at distance and 3° of exophoria at near. Ductions at distance: 4Δ base in to 20Δ base out.

C. C. K.

BOOK NOTICE

SQUINT AND HETEROPHORIA, Dr. R. M. Peckham, published at 11 Monterey Parkway, Rochester, N. Y. 368 pages, 19 diagrams. Price $5.00.

This book is the first of a series of three volumes and deals solely with squint, its etiology and treatment, incorporating various concepts advocated by the author and his associates in various lectures and writings. The first part is devoted to squint, its characteristics and attending problems, such as amblyopia and projection. Refractive factors are also considered. The second part classifies these squints according to the various etiologies and offers prism treatment and relief suggestions. Various new ideas such as that of eyedness and handedness are stressed throughout the book. The author tries to impress upon the reader the importance of forgetting most of the theories we have been taught and advanced by many of the earlier teachers and investigators.

The book is printed on cream color paper with a single narrow page column and wide outside margins. These are special features and make it easier for the reader to follow the contents. Another feature is that there are scattered through the book a number of ruled blank pages which the reader can utilize in making notes while studying the text.

G. L. DuP.

ANNOTATIONS AND SOCIETY PROCEEDINGS

The American Journal of Optometry is the Official Organ of the American Academy of Optometry, as well as the Organ of the Arkansas, Delaware, Indiana, Iowa, Maine, Maryland, Michigan, Minnesota, Nebraska, North Dakota, Oklahoma, Pennsylvania, South Dakota, Vermont and West Virginia Optometric Associations.

The following gentlemen have consented to supply items of interest from their respective sections, acting in the capacity of

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SOUTHEASTERN CONGRESS OF OPTOMETRY

The following notes were taken by Dr. Robert J. Pierce of Winston-Salem, North Carolina, at a recent meeting of the Southeastern Congress of Optometry. Dr. Pierce says in part, that these notes have not been corrected by the lecturers but to the best of his knowledge they are correct.

Barstow's Lecture

Success is a conquest—not a bequest—a conquest of self, not of patients.

One only grows in the social world or in building a practice through his friends.

Personalize the patient.

Believe what the patients do rather than what they say.

Everyone does what he does because he feels like it.

The average person does about 5 minutes thinking a day.

Do not answer what a patient says but what he means.

About ten per cent of the people will try to make you reduce prices. About 20 per cent of the people will wait for bargain sales.

About 70 per cent of the people don't care what they pay for what they get. Patients think less of you if you let them argue you into reducing prices. Don't turn patients adrift when you suspect pathology but send to a specific man and ask him for a report. The

theory of this is if you send him to the man he has been going to he looks on the patient as his instead of your patient.

We are the guardians of the patient's eyes—not their pocketbooks.

Barstow suggests educational pictures on the walls. (Use with discretion.)

Give patients something to talk about by describing examination as you go along and telling them something about their individual case. For instance, you might say, "Mrs. Jones, you have a splendid pupillary reaction to light." Mrs. Jones is very apt to tell her friends that Dr. Kelly said she had such and so, thereby associating Dr. K. with eye examinations in the mind of the friend.

Peckham said some patients do not come back because contact is so short they do not get acquainted.

Barstow said to avoid the danger of mystery in the examination.

Tait's Lectures

Tait tells of some cases that accept minus under atropine and plus without. In one-third of a group of cases, homatropine stimulated accommodation. Trouble of subjective tests is that patients interpret retinal images in the light of his old habit of seeing, which may be all wrong.

Accommodative-convergence and convergence accommodation are definitely related.

Tait thinks ciliary hypertonicity is caused by corneal astigmia, especially of low amounts.

At the near point lenticular astigmia tends to disappear. Dynamic skiametric astigmia agrees with keratometer more than subjective and static do.

Lenticular astigmia may cause cataract.

Dynamic is especially good for pre-school age clinics. Case history ophthalmoscope, keratometer, and dynamic is good routine for pre-school examination.

Forty is the dividing line for Tait's Dynamic. After forty the near correction can be found by working up and stopping at the first neutral reflex. This is the Rx needed at this distance without any deduction.

If physiologic exophoria is low there is more chance of relaxing pseudo myopia and vice versa.

If there is more than 6 or 7 prism diopters of physiologic esophoria, suspect pathology.

Tait never puts prisms on for permanent wear.

By far the most of anyone's patients come to him by being referred, or because they know the optometrist. Therefore, it is extremely important to find out from each patient who, if anyone, referred them, and to thank the referrer. Likewise, it is very important

to become acquainted with as many people as possible.

Tait said that when he does prescribe prisms he arbitrarily puts 2 P. D. base-in over each eye in myopes and 3 P. D. base-in over each eye in hyperopes. (This does not seem to the writer to be a good idea. He prefers Peckham's way of prescribing prisms by the distant outduction recovery. Peckham says that myopes will accept more base-in prisms than hyperopes and the writer's experience bears this out.)

Kraskin on Blood Pressure

Ophthalmoscope does not reveal diseases in their incipiency.

Dr. Neal has found that in presbyopes with hyperphoria of any degree 80% have high blood pressure.

The blood pressure test is essentially simple.

Arterial pulsation in the eye is pathologic.

Normal intra-ocular pressure is 40 mm. of mercury.

If diastolic pressure is under 40 mm. of mercury there is a heart lesion.

If diastolic pressure is over 40 and there is arterial pulsation there is abnormal intra-ocular pressure such as that found in glaucoma.

Insurance companies place great importance on the blood pressure.

Normal heart beat is 72 per minute. The normal heart does enough work in a day to raise a ton 82 feet.

The best way to take blood pressure is the auscultatory method.

A treatise by the U. S. Government called Technical Papers of the Bureau of Standards, No. 352, is about blood pressure and is good. Get it.

The mercury instrument is the best instrument for taking blood pressure.

The best time to take blood pressure is half way between meals.

Nervousness can be detected by rapidity of the pulse.

Don't tell patient the amount of the blood pressure. Later he may have it taken by another examiner and if it doesn't agree with what you told him the other examiner may cast aspersions on your ability to take it.

In rolling up the sleeve to take blood pressure be careful not to bind the arm with the sleeve.

Place the bandage right above the elbow.

Systolic means contraction and diastolic means dilation of the heart.

Pump up slowly in taking blood pressure.

Pump up until you don't feel any pulsation.

Go about 5 mm. above where pulsation stops.

Put tube over the brachial artery. Gradually release until the first sound is heard. This is the systolic pressure.

Dr. Neumueller on Muscles and Fusional Training

The phoria present in a given case is no criterion of the refractive condition. An esophoria caused by a hyperopia may turn into an exhaustion exophoria.

Neumueller said that a hyperphoria with an exophoria of 5 or more is apt to be secondary to the exophoria, and that this is not so in esophoria. Peckham said that a hyperphoria is more apt to be secondary to an esophoria than to an exophoria. (Take your choice.)

Toxic phorias vary and innervational phorias remain fairly constant. This is a good diagnostic point.

Tropias should be divided into two classes—tropias with and without diplopia.

Steps in training phorics without diplopia: First, remove the amblyopia. Second, give motion to the eye. Third, get simultaneous binocular vision or diplopia. Fourth, get single binocular vision.

Visual acuity of amblyopes does not improve regularly but in jumps. This is a good thing to remember when several treatments do not improve vision.

Make the bad eye work in treating amblyopia.

Vision is learned by feeling with the hands.

Nutritional disturbances affect blue fields first.

"Presbyopia is an untouched field" (Neumueller).

Correct the presbyope for 16 or 17 inches.

High exophoria at near is common to presbyopes. Too much add will make the new presbyope have discomfort by suddenly increasing his near exophoria. Give the new presbyope two or three sittings of prism exercises to build up near ductions. Sometimes near induction can be built up quicker with base-in exercises than with base-out.

It is often a good idea to decenter segments more than the difference between the distant and near p. d. to give a base-in effect at near.

"It is a fallacy that the base-down effect of large segments makes the esophore relax his convergence because he looks higher than the wearer of small segments. This is because the base-down effect is so small."

All prism exercises are shock systems.

Fusional urge is stronger than mere duction.

Sterling on Bifocals

The bifocal user looks 6 to 8 mm. below the optical center of the distance lens the most. For this reason it is wise to figure the size of the segment so that there will be a minimum of vertical prism power at this point.

Formula for calculating the size of segment to have no vertical prism at a point 6 mm. below distance center:

$$6\left(\frac{\text{Distance power at } 90^\circ}{\text{add}}+1\right)=\text{distance in mm. that center of}$$

segment is below distance center to have no vertical prism at a point 6 mm. below distance centers.

If add is about the same as the distance power a 22 mm. segment is indicated.

If add is less than distance power the segment should be larger than 22 mm.

If add is more than distance power the segment should be smaller than 22 mm.

Use different size segments in anisometropia to balance up the vertical prismatic effect at a predetermined point (6 or 8 mm. below distance centers). There is a formula for this, but it is a fairly simple matter to calculate it without using a formula by first calculating difference in the vertical prism effect of the distance powers only at a point 8 mm. (Sterling said he thought 8 is better than 6) below the distance centers. The lens that has the most base-up effect from the distance power will need the large segment to give a prism base-down effect.

Illustrative case of figuring segments size to compensate for vertical prism effect of anisometropic correction:

Right + 1.00 S. add 2.00.

Left + 2.25 S. add 2.00.

The difference in the distance powers is 1.25 S. At a point 8 mm. below distance centers there will be .8 times 1.25, which equals 1.00 prism diopters of vertical prism base-up in the left eye. The left segment must have center lower than the right to compensate for this. The formula for prismatic power is: Distance from optical center in centimeters times the dioptric power equals the prismatic power. Let X equal the distance in centimeters. Then:

X times 2.00 equals 1.00.

2X equals 1.00.

X equals .5 centimeters, or 5 mm.

Therefore the center of the segment in the left segment must be 5 mm. below that in the right segment. If the right lens is made up in a Kryptok with a 22 mm. segment, or a segment of 11 mm. radius, the left should be made up in a segment of 16 mm. radius, probably an Ultex. This amount of anisometropia (1.25 S.) isn't at all unusual. We all know that 1.00 prism diopter of hyperphoria is apt to cause discomfort, yet we have been placidly creating an artificial hyper-

(Continued on page X)

Arista

THE MOUNTING

BY the very act of choosing Orthogons, the patient lists himself as appreciating what is newer and better in eyewear. Automatically, then, you please him by offering Arista Natural Gold Mountings—the new color in the new rimless vogue. The growing volume of Arista Rimless demonstrates increasing appreciation of rimless advantages—including that universal preference, inconspicuous eyewear. Current Mountings in Arista are of proved popularity—Sovereign, Clingswell, Hy-Clingswell. New rimless designs, also in Arista Natural Gold are now in preparation.

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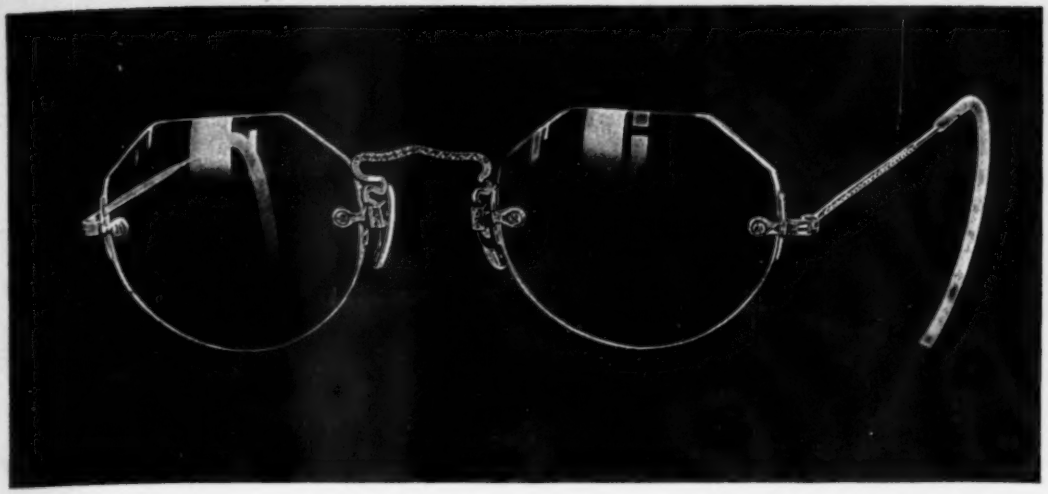

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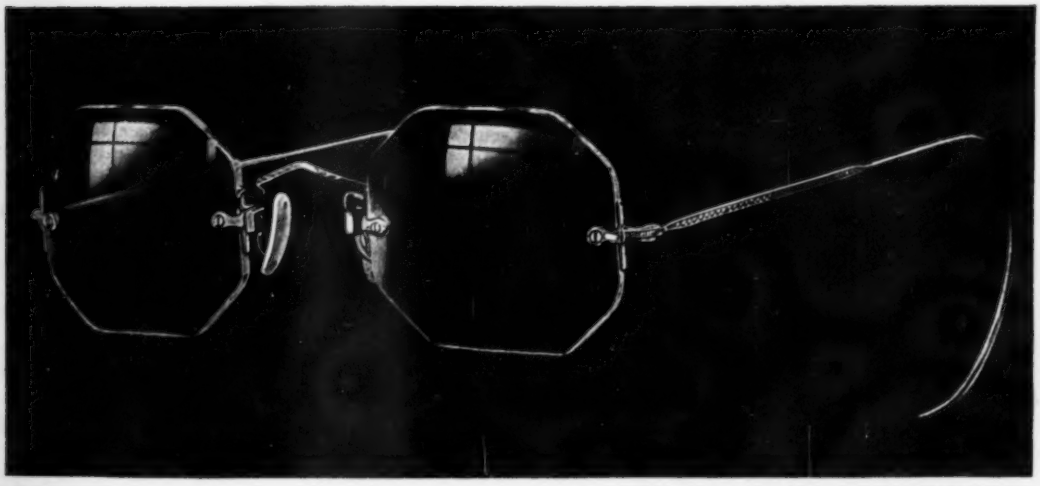

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(Continued from page 323)

phoria of this at the reading point of the bifocals we prescribe. Using different sized segments will correct this error.

(To Be Continued)

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BACK COPIES WANTED

Dr. W. A. Springborg, 506 Capitol Bank Bldg., Lansing, Michigan, is anxious to secure the following copies of the American Journal of Optometry: Vol. 7—Nos. 2, 3, 4, 6, 7, 9, 11, and Vol. 8—No. 2. Dr. Springborg is willing to purchase these copies and any reader who can supply him with them should communicate directly with Dr. Springborg.

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